Tendon and Muscle Imaging in Sports

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Summary

Tendon and muscle lesions appear in manifold forms clinically as well as on magnetic resonance or ultrasound images. Tendinous abnormalities may be classified and staged with respect to their biomechanical role in a kinetic chain by using the concept of the tendon overuse syndrome (TOS). In the first phase, painful functional impairment of movements occurs without any morphological changes. In the second stage, abnormalities of the gliding tissue in the form of bursitis, tendovaginitis or peritendinitis are observed. In the third stage, such lesions are followed by degenerative changes of the tendon itself. Often, they present more clearly than during the early forms of TOS and three types of tendon degeneration can be differentiated: tendinosis at distinct points along the course of the tendon, fibroostosis at the tendon insertion and compression or impingement syndromes. Rupture of fibres following tendinosis may be considered as the last or fourth stage of TOS.

Introduction

Ruptures of tendons or muscles may occur because of direct trauma from blows in contact sports or from perforating wounds. More commonly observed are ruptures because of indirect trauma at points of greatest weakness within a kinetic chain, so-called critical zones: repetitive overuse causes degeneration with inflammation at these zones and eventually may result in a rupture (1, 2). Thus, a torn tendon or muscle should be regarded rather as the final stage of a continuously progressive overuse syndrome than as a sudden event as it is experienced by most patients in the form of a striking blow (similar feelings to those reported about the ancient hero Achilles hit by Apollo's arrow are today described by contemporary high-ranking athletes). The incidence of tendon lesions has been rising dramatically during the last decades and seems to be associated with modern lifestyle problems (3). Diagnostic imaging offers new insights into the wide variability of overuse syndromes and injury of tendons and muscles.

The development of overuse syndromes depends on the amount of mechanical stress applied on a kinetic chain and on its resistance against such stress. The latter is because of genetic and constitutional parameters, gender (ruptures of the Achilles tendon almost always occur in males) and a higher vulnerability in certain periods of lifetime (vulnerable phases): in adolescents the tendon insertions are specifically prone to stress (apophysitis), in young adults ruptures of muscles or at musculo-tendinous junctions are observed with higher frequency and during the third and fourth decades of life and later various forms of tendon degeneration and rupture may occur.

Most of these lesions are associated with various sporting activities and some of them with occupational stress. Depending on their anatomical location and their severity, tendon and muscle lesions appear in manifold forms on magnetic resonance (MR) or ultrasound (US) images (Table 1). They may, however, be classified and staged following standardized rules with respect to their biomechanical role in a kinetic chain with respect to the concept of the tendon overuse syndrome (TOS) (4).

Indications for imaging

Sports-related referrals should be specified as following (5).

In case of a tendon overuse, a referral for imaging generally bases on pain with or without swelling or on functional impairment because of a mismatch between applied forces and tensile strength. Diagnostic imaging should be performed to describe the type and the stage of tendon overuse with the aim to initiate proper therapy. Conclusions to estimate the risk of a rupture may be drawn from the imaging appearance but this is still under debate (2, 6, 7). The risk of a rupture is determined by intrinsic and extrinsic parameters (Table 2) and especially with US and MR imaging (MRI), it is possible to analyse many of the intrinsic factors in detail (8).

In traumatic events with involvement of muscles, contusions because of direct forces or strains because of indirect forces should be investigated in high-ranking athletes for supporting the decision of defining the return to play. Stiff muscles or low-degree strains are commonly

■ **Table 1:** Common tendon overuse syndromes

Syndrome	Localization	Aetiology
Upper extremity		
Impingement (subacromial or postero-superior)	Supra- and infraspinatus tendons	Tendon compression and/or increased traction forces Extrinsic impingement (narrowing of subacromial space or repetitive overhead motions of arm)
Rotator cuff fibroostosis (enthesopathy), sometimes as partial supraspinatus tendon avulsion (PASTA) lesion		Intrinsic impingement (structural changes of rotator cuff, i.e. abuse of anabolic steroids)
Osteochondral lesion of humerus		
Subcoracoidal impingement	Subscapularis tendon	Often post-traumatic compression of coracoid process
Superior labrum anteroposterior (SLAP)-lesion	Long biceps tendon	Injury of the labral biceps tendon anchor
Rotator cuff interval lesions, microinstability Hidden lesions	Long biceps tendon	Degeneration or rupture at the level of rotator cuff interval or within the osteofibrous channel between greater and lesser tubercles (biceps tendon pulley) Subluxation or luxation of biceps tendon may be associated with subscapularis tendon rupture
Pectoralis tendon tear	Pectoralis major tendon	
Ulnar epicondylitis (Golfer's or thrower's elbow)	Flexor carpi ulnaris and other flexor tendons	Micro- and macroruptures with inflammation Traction apophysitis in adolescents
Little leaguer's elbow		
Radial epicondylitis (Tennis elbow)	Tendon of extensor carpi radialis brevis muscle (ECRB)	Micro- and macroruptures with inflammation
Distal biceps tendon lesions	Tendon of biceps brachii muscle	Bursitis, tendinosis or rupture following weight-lifting or throwing
Thrower's elbow	Distal triceps tendon	Tendinosis with bursitis because of dorsal or dorsomedial impingement
DeQuervain's disease (housewife's wrist, oarsman's wrist) intersection syndrome	Tendons of abductor pollicis longus and extensor pollicis brevis muscles	Tendovaginitis Intersection syndrome: involvement of same tendons with extensor radialis longus and brevis, but more proximal
Climber's finger Trigger finger	Flexor tendons of fingers	Anular ligament lesions (especially A2, A3) with tendovaginitis and tendinosis Stenosing tendovaginitis
Extensor carpi ulnaris tendon tendonitis or luxation	Tendon of extensor carpi ulnaris muscle	Overuse within osteofibrous tunnel along the ulnar styloid process
Lower extremity		
Tractus syndrome (trochanteric bursitis with or without lateral snapping hip)	Iliotibial tract	Friction at greater trochanter
Iliopsoas bursitis (with or without medial snapping hip)	Insertion of iliopsoas tendon	Friction with or without subluxation
Adductor muscle tendinosis or bursitis	Ischiopubic origin of adductor tendons	Repetitiv abrupt tension of muscles
Rectus femoris overuse	M. rectus femoris	Tendinosis or rupture of proximal musculo-tendinous junction (of caput reflexum)
Hamstring syndrome	Semitendinosus, semimembranosus and biceps femoris tendons	Shortening of hamstring muscle bellies
Proximal patellar spur	Insertion of quadriceps tendon	Degeneration with traction spur
Runner's knee (iliotibial band friction syndrome)	Tractus iliotibialis	Friction oedema between iliotibial tract and lateral femoral epicondyle
Jumper's knee (apex patellae syndrome)	Patellar tendon	Degeneration with inflammation at origin of tendon
Popliteus tendinosis or bursitis	Popliteus tendon	Friction with effusion in popliteal recess
M. Osgood–Schlatter	Patellar tendon	Traction tendinitis with deep infrapatellar bursitis
M. Sinding–Larssen–Johannson	Apex patellae	Adolescent traction tendinitis of patellar tendon with elongation of apex patellae
Achillodynia	Achilles tendon	Hindfoot hyperpronation
Posterior calcaneal spur	Achilles tendon	Vermehrte Zugwirkung am Ansatzpunkt der Achillessehne
Apophysitis calcanei (Sever's diseases)	Achilles tendon	Fragmentation of calcaneal apophysis because of increased tensile strength of tendon
Haglund's heel	Achilles tendon	Compression syndrome of tendon between posterior aspect of calcaneus and shoe
Plantar fasciitis (anterior calcaneal spur)	Plantar aponeurosis	Traction osteophyte with thickening at medial tubercle of calcaneus

■ **Table 1:** Continued

Syndrome	Localization	Aetiology
Posticus lesion	Tibialis posterior tendon	Increased axial load of tendon because of flattening of plantar dome
Peroneal tendon lesion (jogger's foot)	Peroneal tendons	Subluxation of tendons within the osteofibrous channel along distal fibula
Ventral ankle tendon lesions	Tibialis anterior and other long extensor tendons	Friction of tendons beneath extensor retinaculum
Dancer's heel, soccer's heel	Flexor digitorum longus tendon	Friction adjacent to os trigonum

■ **Table 2:** Extrinsic and intrinsic factors that influence the development of tendon overuse syndromes

Intrinsic factors
Malalignment
Leg length discrepancy
Femoral anteversion
Joint alignment abnormalities (varus, valgus, other)
Foot abnormalities (flat or cavus foot)
Muscle tension imbalance
Variants in the origin, course or insertion of tendons
Shallow osseous canal
Accessory muscle
Accessory or atypically formed bone
Hypovascularization of tendon tissue
Underlying systemic metabolic or inflammatory disease
Psychological factors or psychiatric disease
Personal attributes
Gender
Age
Weight and height
Growth
Extrinsic factors
Training errors
Excessive training
Abrupt changes in intensity of training
Unskilled athlete
Doping (testosterone)
Improper footwear or clothing
Environmental factors
Hard or uneven ground

observed in athletes prior to a competition and the pain associated with them is at least partly driven by psychosomatic moments. There is no indication for imaging unless signs of a nerve injury (so-called 'red flags') are present (as in the form of 'burners' and 'stingers' indicating a brachial plexus lesion because of a cervical spine injury).

Ultrasound is the modality of choice to detect abnormalities of all superficially located tendons and muscles (5). With MRI, more deeply located structures should be investigated. A further potential of MRI is its ability to detect structural abnormalities in more detail, surrounding oedema and associated abnormalities within a kinetic chain. Conventional radiographs should not be selected as primary investigations but rather for answering specific questions related to malalignment calcifications and osseous abnormalities impairing the gliding mechanism. Computed tomography (CT) may be of use in case of trauma or to analyse calcifications in detail. Special radiological techniques like tenography with instillation of contrast media in a tendon sheath have been almost completely replaced by cross-sectional imaging.

Investigation

High-resolution techniques should be applied to document the internal structures of collagen and muscle fibres as well as of their gliding tissues (9, 10).

Ultrasound investigations are generally performed with transducer frequencies of more than 10 MHz (11). Panoramic or extended view modes are helpful to display the whole course of a tendon. Doppler examinations may be useful to detect inflammatory hypervascularization within the generally hypovascular and bradytrophic tendon tissue.

Magnetic resonance images should be obtained in at least two plains parallel and perpendicular to the course of a tendon with T1-weighted or intermediate-weighted and with fat-suppressed T2-weighted sequences. With high-resolution imaging techniques (intermediate-weighted or gradient-echo imaging) the internal structures of many bigger and smaller tendons are displayed readily. Kinematic MRI may, despite the limited clinical experience with this technique, become a useful tool in evaluating certain pathological conditions like subluxation of the peroneal tendons or complex functional impairment of the hindfoot (12).

Computed tomography data sets, gained by using multislice–multidetector technology, should be routinely reformatted in three image planes. With modern software equipment, additional reconstructed 3D-volume images may be used to display tendons and muscles as the different Hounsfield unit values of these structures are discernible (13).

Interpretation

Normal imaging anatomy

On high-resolution US images, normal tendons consist of hypoechoic collagen fibre bundles that appear hypointense with MRI (14, 15). They are intermingled between the internal peritendineal tissue that is sonographically hyperechoic and with MRI hyperintense on all sequences except

on fat-suppressed images. The normal tendon size is varying; on the foot the general agreement is that tendons are normal if smaller than 4 mm except of the Achilles tendon and the plantar fascia that should not exceed more than 6 mm. As tendons are anisotropic structures, focal signal inhomogeneities because of a magic angle artefact on T1-weighted images have to be interpreted with great caution.

Signs of tendon and muscle pathology

Malalignment

Frequent entities being in close anatomical and biomechanical context with a higher risk of degeneration are the hyperpronation of the hindfoot, flattening of the plantar dome and the different forms of shoulder impingement and of snapping hip.

Structural abnormalities of tendons

Tendon degeneration and rupture generally manifest in the form of distinct patterns that are closely related to the type of biomechanical stress and the duration as well as the severity of the disease. Such, four stages of the TOS may be described (4):

- In the first phase of TOS, painful functional impairment of movements occurs without any morphological changes.
- In the second stage, abnormalities of the gliding tissue in the form of bursitis, tendovaginitis or peritendinitis are observed (Fig. 1). Chronic inflammation of the tendon sheath may lead to stenosing tenosynovitis with fibrosis and tendon entrapment.
- In the third stage, such lesions are followed by degenerative changes of the tendon itself. Often, they present more clearly than during the early forms of TOS and three types of tendon degeneration can be differentiated: tendinosis at distinct points along the course of the tendon, fibroostosis at the tendon insertion and compression or impingement syndromes (Fig. 2).
- Rupture of fibres following tendinosis may be considered as the last or fourth stage of TOS (Fig. 3).

In tendinosis, the most distinctive sign is a fusiform swelling in the body of a tendon. Especially, on axial images they manifest as irregular, reticular and hyperintense foci, which in most cases begin to develop at certain points of increased stress. Abnormalities of cross-links among collagen fibres are accused to play a certain role in the development of instability, which eventually may end in a rupture (16).

Various theories have been established, which may be grouped in those in which a localized loss of blood supply ('microinfarctions') and those in which increased stress ('microruptures') are attributed to explain the aetiology of such lesions.

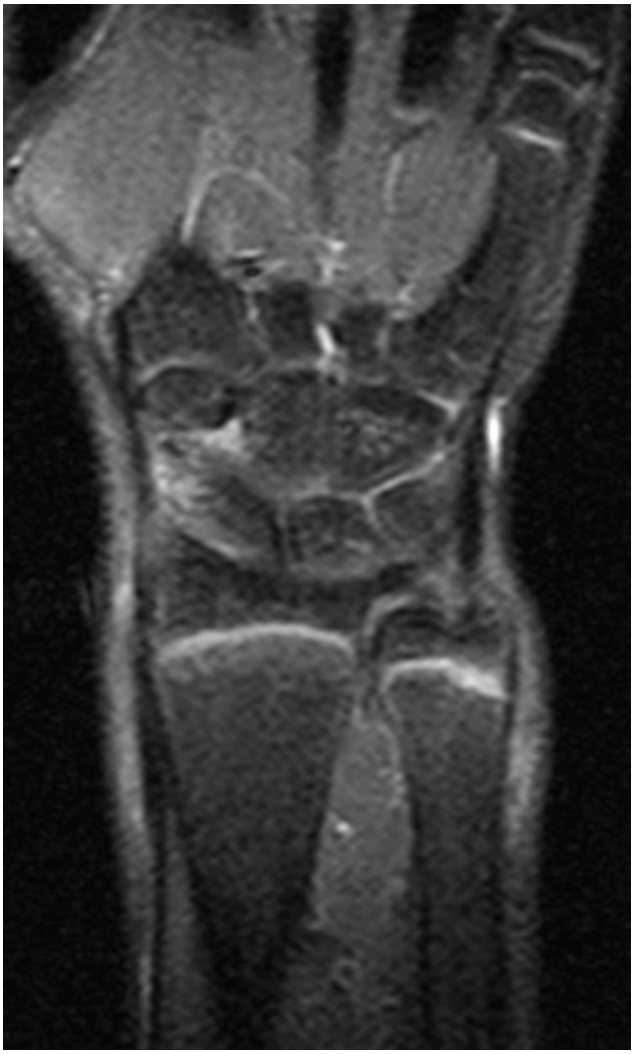
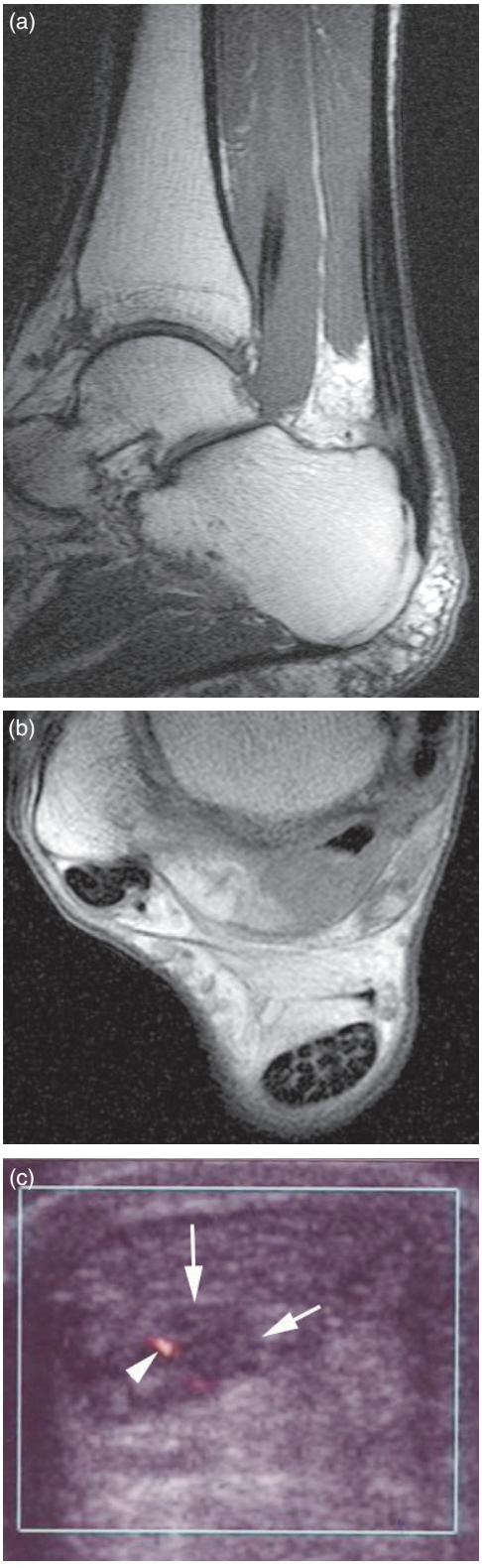


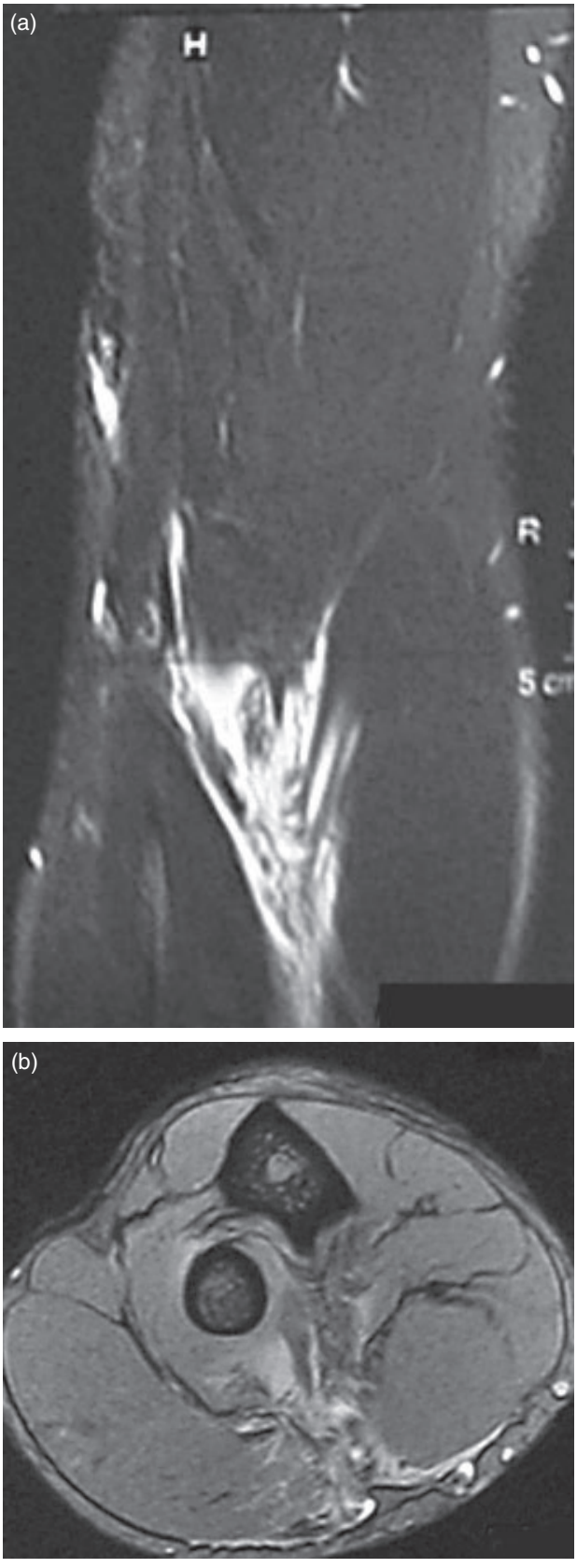
Fig. 1. Tendon overuse syndrome stage 2 in a young female gymnast with oedema surrounding the radial and ulnar tendons of the wrist. Associated findings are effusion around scaphoid and hyperintense signal alteration of distal ulnar physis.

Abnormalities at the tendon insertion generally manifest with traction osteophytes, i.e. spurs or fibroostosis. In adolescents, juvenile apophysitis or a periosteal desmoid may be observed at various sites, Osgood–Schlatter's disease of the patellar ligament and the adjacent deep infrapatellar bursa, Sever's disease of the Achilles tendon or the little leaguer's elbow are common forms (17).

Signs of rupture reflect the discontinuity of tendon fibres and are often associated with haemorrhage. A tendinous gap can be detected and its extent described in detail (14). Partial tendon tears can be defined as linear or focal regions of increased signal and thickening of fibres without a tendinous gap. In many cases, it may be difficult to distinguish areas of tendinosis from chronic intrasubstance tears without documenting either discontinuity in tendon fibres or discrete hyperintense signal intensity on T2-weighted or short-time inversion recovery images with



■ **Fig. 2.** Tendon overuse syndrome stage 3 in a male jogger with (**a**) marked thickening of Achilles tendon and swelling of deep Achilles tendon bursa, (**b**) thickened internal peritendineal as visible on axial images, and (**c**) moderate hypervascularization visible with power Doppler ultrasound at level of bursa.



■ **Fig. 3.** Tendon overuse syndrome stage 4 in the form of a ruptured distal biceps tendon after years of overuse. (**a**) Fluid accumulation at insertion site, which (**b**) follows the course of the tendon and its adjacent bursa.

partial tears. Subacute haemorrhage generates high-signal intensity on T1-weighted images.

Structural abnormalities of muscles

Muscle strains may be graded as:

- first degree strains with microruptures (<5% of muscle diameter) and signs of 'feathered haematoma',
- second degree strains, i.e. partial muscle ruptures, and
- third degree strains (complete ruptures) with retracted fibres (18).

The 'critical zones' for muscle ruptures are located in the large muscles of the lower extremity running across more than one joint. Specific forms of muscle ruptures are:

- Hernia, which may be visualized as lump by using dynamic ultrasound.
- Acute or chronic compartment syndrome with or without soft tissue oedema.
- Sequels of entrapment neuropathy with atrophy or fatty transformation of the muscle tissue.
- Traumatic myositis ossificans manifests in three stages: acute or pseudoinflammatory, subacute or pseudotumourous and chronic. Extensive muscle oedema at the beginning resolves progressively and ends with calcification and final ossification, in which a typical 'zonal phenomenon' may be observed.
- The inflammatory pseudotumour is a nodular inflammation following a muscle rupture and is in most cases observed within the rectus femoris muscle.

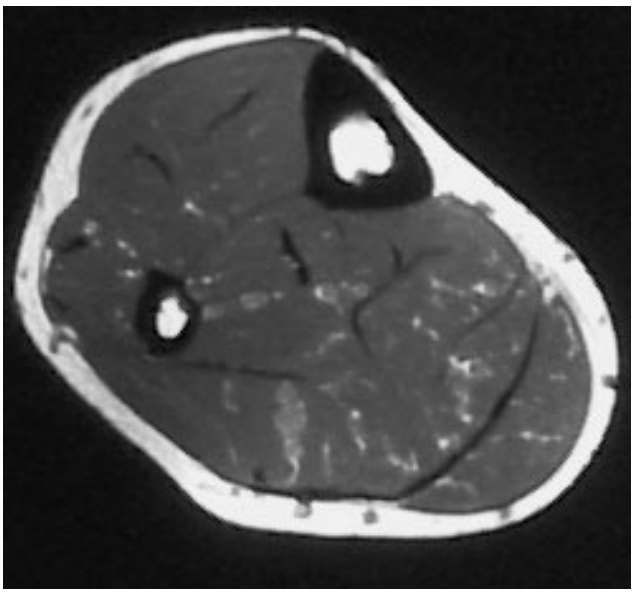
Findings in adjacent joints and bones

Many form of TOS are associated with accompanying abnormalities of the adjacent joints or bones. Joint effusions are commonly observed in major joints. Bone marrow oedema may result from repetitive contusions or from traction movements within the subchondral bone. A typical location of oedema within the cancellous bone is near osteofibrous tunnels of large tendons. In more severe cases, oedema forms in some distance apart from joints is indicative of stress or insufficiency fractures.

Differential diagnosis

Because of the inherently high biomechanical load on tendon or muscle fibres findings of degeneration dominate the clinical and the radiological appearance not only in degenerative and traumatic, but also in many other forms of tendon or muscle diseases.

Soft tissue tumours may be associated with bleeding and are, therefore, difficult to be differentiated from traumatic haematomas (Fig. 4). Tumours of the tendons are rare except of ganglia that are commonly observed in the dorsal tendon sheaths of the foot. Giant cell tumour of the tendon sheath is reported to occur typically in the ankle and the foot. Generally, all neoplasms that originate from the synovium may be observed in tendon sheaths. A periosteal desmoid, typically located at the distal femur, may be difficult to be differentiated from early forms of osteosarcoma.



■ **Fig. 4.** Soccer player with subacute haematoma at his calf. A clinically suspected soft tissue tumour could be excluded.

Inflammation may be because of infections (abscess formation after penetrating trauma), autoimmunological disorders (Reiter's disease and other forms of reactive arthritis, early rheumatoid arthritis or others) or may develop as post-traumatic synovitis within tendon sheaths.

The most common metabolic disorders manifesting in tendon tissue is hydroxyapatite pyrophosphate arthropathy (calcific tendonitis) of the rotator cuff or the gluteal muscle tendons, whereas other crystal-induced musculoskeletal diseases (calcium pyrophosphate dehydrate deposition disease, gout) are less commonly encountered. The effect of corticosteroids from local or systemic application has been described to cause tissue necrosis (5). Certain forms of hyperlipoproteinaemia are associated with structural tendon abnormalities. Recently, the role of anabolic drugs has been stressed in the development of tendon necrosis.

Diagnosis

Diagnostic imaging of tendons should be performed with the aim to clarify the reasons of a painful overuse syndrome. Compared with muscles and bones, the bradytrophic tendons have a relatively long time of adaptation and regeneration with consequences in training and living habits. Especially in the field of sports medicine, the

imaging findings have to be correlated with individual patterns of overuse in order to stop the progressive course of tendon disease from functional impairment, which has a good prognosis of healing, to manifest rupture. The analysis of complex overuse syndromes may be helpful to aid and specify treatment options. Sonographically guided antiphlogistic injections are of help especially at complex anatomical sites (19). Muscle disorders should be investigated with US or MRI to determine the type and the extent of traumatic events and to differentiate them from cystic or solid neoplasms.

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